

# Moderate dietary fat consumption as a risk factor for ischemic heart disease in a population with a low fat intake: a case-control study in Korean men<sup>1-3</sup>

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## ABSTRACT

**Background:** Dietary fat intake is associated with the incidence of ischemic heart disease (IHD) in Western countries. In populations in which both the average dietary fat consumption and the incidence of IHD are lower than in Western countries, the association of dietary fat intake with IHD incidence remains unknown.

**Objective:** We conducted a case-control study to examine the association of dietary fat with IHD incidence in Korean men.

**Design:** The case group consisted of 108 patients with electrocardiogram-confirmed myocardial infarction or angiographically confirmed ( $\geq 50\%$  stenosis) IHD who were admitted to a university teaching hospital in Seoul, Republic of Korea. The controls were 142 age-matched patients admitted to the departments of ophthalmology and orthopedic surgery at the same hospital. Dietary fat intake was assessed by a nutritionist using a semiquantitative food-frequency questionnaire. Body mass index (BMI), cigarette use, alcohol intake, exercise, and history of disease were determined during an interview and examination.

**Results:** In a univariate analysis, the mean percentages of energy from total fat, saturated fatty acids, and monounsaturated fatty acids were significantly higher in the cases than in the controls. BMI, smoking, and a history of hypertension were associated with the occurrence of IHD. In multiple logistic analyses, total fat intake was a significant risk factor (odds ratio: 1.08 for 1% of energy intake; 95% CI: 1.02, 1.14) after adjustment for BMI and smoking.

**Conclusion:** In a population with a relatively low fat intake (19% of energy intake), a moderate increase in total fat intake may be a risk factor for IHD. *Am J Clin Nutr* 2001;73:722-7.

**KEY WORDS** Ischemic heart disease, dietary fat intake, Republic of Korea, men, case-control study

## INTRODUCTION

In Western countries, the results of epidemiologic studies such as the Western Electric Study (1), the Zutphen Study (2), the Honolulu Heart Program (3), the Puerto Rico Heart Health Program (4), the Ireland-Boston Diet-Heart Study (5), and the Seven Countries Study (6) indicate that dietary factors such as total fat, saturated fatty acids (SFAs), polyunsaturated fatty acids (PUFAs), *n*-3 series fatty acids, and cholesterol are associated with the incidence of ischemic heart disease (IHD).

In these countries, total fat intakes are  $\approx 35$ –40% of energy and current guidelines recommend that  $< 30\%$  of energy come from fat to prevent fat-related diseases (1–6, 7). In Japan the new recommended dietary fat allowance is 20–25% of energy intake (7).

In China dietary fat intake increased from 15.9% of energy in 1982 to 21.1% in 1990; IHD incidence and mortality also increased (8). In China as well as in Japan, Campbell and Junshi (9) and Wenxun et al (10) reported associations between diet, metabolic indicators of lipid status, and IHD incidence and mortality. Nevertheless, uncertainty remains about the optimal fat intake for the prevention of IHD in populations with relatively low fat intakes.

Mortality from IHD among Koreans in 1996 was estimated as 14 per 100,000 persons. Even though this rate is low compared with that in Western countries, it has increased 5- to 6-fold during the past decade (11). Paralleling the recent economic development in Korea, the average fat intake of Koreans increased during the same period: from 14% of energy in 1986 to 19% of energy in 1997 (12). This increased dietary intake may affect the incidence of IHD.

Studies in Korea that addressed the effects of risk factors on IHD incidence identified the same risk factors as did studies conducted in Western countries (13–18). Smoking, hypertension, hyperlipidemia, diabetes mellitus, and obesity seem to be related

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to the incidence of IHD. However, in most studies in Korea, nutritional risk factors were not studied or only partial outcomes were reported (19–21). In this study, we investigated the association of fat intake with the incidence of IHD in Korean men.

## SUBJECTS AND METHODS

### Subjects

From October 1995 through July 1996, cases and controls were selected from a university teaching hospital in Seoul. The cases were 108 male patients with a first acute myocardial infarction (50 patients) or a new diagnosis of angina pectoris (58 patients) who were admitted to the Cardiovascular Center. The inclusion criteria for the cases were 1) age  $\leq 75$  y, 2) onset of symptoms within 1 mo of admission, 3) acute myocardial infarction confirmed by electrocardiogram and cardiac enzyme measurement, and 4) angina pectoris confirmed by coronary angiogram ( $\geq 50\%$  occlusion in  $\geq 1$  of 3 main coronary arteries).

One hundred forty-two controls, matched by age within 10 y, were selected from male patients admitted to the departments of ophthalmology (69 patients) and orthopedic surgery (73 patients). The inclusion criteria for the controls were 1) normal electrocardiograms confirmed when the patients were admitted to the hospital and 2) no history of myocardial infarction or angina pectoris. Also, we excluded patients aged  $> 75$  y and those who had experienced any changes in their usual dietary pattern within the past year.

### Methods

A trained interviewer interviewed the cases and controls in the hospital. Variables measured in this study were sociodemographic characteristics (age, marital status, education, and income), cigarette smoking status, alcohol intake, exercise, and history of disease (stroke, hypertension, hyperlipidemia, and diabetes mellitus). Height and weight were measured at hospital admission and body mass index (BMI) was calculated by dividing weight (kg) by height squared ( $m^2$ ). Leisure-time physical exercise was measured by asking the type and frequency of exercise performed weekly. Cigarette smoking history was assessed by questions about current or past use of cigarettes; to be considered an exsmoker, a patient had to report quitting smoking  $\geq 1$  y before the time of admission. Questions about alcohol intake assessed the frequency of drinking per week and the amount of alcohol consumed.

Dietary data were obtained by a nutritionist using a semiquantitative food-frequency questionnaire (FFQ). We developed our own FFQ that listed 83 food items generally consumed by Koreans daily and that contain substantial amounts of total fat and fatty acids. Each subject was asked to report the usual frequency of consumption and the usual portion size during the past year. The frequency of consumption was measured on a 9-grade scale: never, 1 time/mo, 2 times/mo, 1 time/wk, 3 times/wk, 5 times/wk, 1 time/d, 2 times/d, and 3 times/d. The questionnaire also included a specific question about the type of fat used in cooking. This information was used in the recipes of the mixed dishes. During the interview, food models and reference utensils were shown to the subjects so that they could estimate portion sizes. The participants were interviewed within a week of admission to the hospital and were asked to describe their usual dietary patterns before diagnosis of any known coronary disease. When it was apparent that a patient had substantially changed his usual dietary pattern within the past year, we excluded him from the analysis.

Nutrient intake based on this information was calculated from food-composition tables in Korea, and fatty acids and cholesterol were calculated according to other published data (22–24). Daily nutrient intake was calculated by multiplying the nutrient content of the specified portion of each food item by the frequency of its daily consumption and summing over all items. The dietary information thus calculated included each person's usual daily intake of energy, total fat, protein, carbohydrate, SFAs, monounsaturated fatty acid (MUFAs), and PUFAs.

The FFQ was validated in a pilot study carried out with 31 men who visited the hospital for health screening. The FFQ was compared with a 3-d diet record and was shown to provide a reasonable measure of both total and specific types of fat; the unadjusted correlations between the FFQ and the diet record were 0.60 for energy, 0.43 for total fat, and 0.40 for SFAs.

### Statistical analysis

For the present analysis, we categorized variables as follows: years of education =  $< 10$ , 10–12, or  $\geq 13$ ; marital status = married or separated-unmarried; monthly income =  $< \$1000$ ,  $\$1000$ – $\$1999$ ,  $\$2000$ – $\$2999$ , or  $\geq \$3000$ ; occupation = white collar, blue collar, or other; smoking status = current smoker, exsmoker, or never smoked; alcohol intake = current drinker, exdrinker, or nondrinker; leisure-time physical exercise = every day, 3–6 times/wk, 1–2 times/wk, no regular exercise, or none; and BMI =  $< 25$  or  $\geq 25$ .

We compared age, BMI, and the mean intake of specific nutrients in the cases and controls by use of two-sample *t* tests. We used chi-square tests to compare categorical variables. Statistical significance ( $P < 0.05$ ) was the guideline for selecting independent variables to be included in the final logistic regression model. No significant interactions were found between smoking status and dietary fat, between smoking status and BMI, or between total fat intake and BMI (data not shown). Therefore, according to the parsimonious principle, we excluded the interaction terms from the logistic regression model. BMI, cigarette smoking, energy intake, and dietary fat intake were included in the final logistic regression model. Data analysis was performed with SAS software (version 6.12; SAS Institute Inc, Cary, NC).

## RESULTS

The distributions of the cases and controls by categories of age, BMI, marital status, education, monthly income, cigarette smoking status, alcohol intake, physical exercise, and history of disease are shown in **Table 1**. There were no significant differences between the cases and controls in the distributions of age, marital status, and educational levels, but BMI was significantly higher in the cases than in the controls. Monthly income was slightly higher in the cases than in the controls, but not significantly so.

The proportion of current smokers was significantly higher in the cases (65%) than in the controls (49%). The frequency of alcohol consumption and the frequency of exercise did not differ significantly between the cases and controls.

Significantly more cases than controls had a history of hypertension or hyperlipidemia. Thirty eight percent and 27% of the cases had been previously diagnosed with hypertension or hyperlipidemia, respectively. For history of hypertension, the proportion of respondents with an unknown history was small and this is unlikely to have affected the difference between the cases and controls. For history of hyperlipidemia, however, the proportion of patients with an unknown history was large.



**TABLE 1**

General characteristics of the cases and controls

Variable	Cases (n = 108)	Controls (n = 142)
n (%)		
Age (y)		
<50	38 (35.2)	52 (36.6)
50–59	37 (34.3)	49 (34.5)
≥60	33 (30.6)	41 (28.9)
Body mass index (kg/m <sup>2</sup> ) <sup>1</sup>		
<25	54 (50.0)	97 (68.3)
≥25	54 (50.0)	45 (31.7)
Marital status		
Married	107 (99.1)	139 (97.9)
Single or widowed	1 (0.9)	3 (2.1)
Education (y)		
≤9	28 (25.9)	56 (39.4)
10–12	44 (40.7)	45 (31.7)
≥13	36 (33.3)	41 (28.9)
Monthly income <sup>2</sup>		
<\$1000	21 (20.2)	46 (33.3)
\$1000–1999	42 (40.4)	50 (36.2)
\$1999–2999	21 (20.2)	19 (13.8)
≥\$3000	20 (19.2)	23 (16.7)
Smoking status <sup>3</sup>		
>1 pack/d	28 (25.9)	10 (7.0)
≤1 pack/d	42 (38.9)	59 (41.6)
Exsmoker	28 (25.9)	40 (28.2)
Never smoked	10 (9.3)	33 (23.2)
Alcohol intake <sup>4</sup>		
Drinker	64 (59.8)	88 (62.0)
Exdrinker	22 (20.6)	24 (16.9)
Nondrinker	21 (19.6)	30 (21.1)
Exercise <sup>5</sup>		
Every day	18 (16.8)	16 (11.4)
3–6 times/wk	3 (2.8)	11 (7.9)
1–2 times/wk	12 (11.2)	18 (12.9)
Not regularly	74 (69.2)	95 (66.2)
History of hypertension <sup>3</sup>		
Yes	41 (38.0)	20 (14.1)
No	59 (54.6)	103 (72.5)
Unknown	8 (7.4)	19 (13.4)
History of hyperlipidemia <sup>6</sup>		
Yes	29 (26.8)	18 (12.7)
No	42 (38.9)	76 (53.5)
Unknown	37 (34.3)	48 (33.8)
History of stroke		
Yes	3 (2.8)	1 (0.7)
No	105 (97.2)	141 (99.3)
History of diabetes		
Yes	16 (14.8)	15 (10.6)
No	92 (85.2)	127 (89.4)

<sup>1,3,6</sup>Significant difference between cases and controls (chi-square test):<sup>1</sup>P = 0.003, <sup>3</sup>P = 0.001, <sup>6</sup>P = 0.009.<sup>2</sup>The number of missing subjects was 4 for cases, 4 for controls.<sup>4</sup>The number of missing subjects was 1 for cases.<sup>5</sup>The number of missing subjects was 1 for cases, 2 for controls.

Crude nutrient intakes and nutrient intakes as a percentage of total energy are shown in **Table 2**. The average daily intake of energy was 9873.6 kJ in the cases and 9302.9 kJ in the controls. When macronutrient intake was expressed as a percentage of total energy intake, the total fat intakes of the cases and controls were 22.4% and 19.9%, respectively. Total fat intake was signifi-

cantly higher and carbohydrate intake significantly lower in the cases than in the controls.

The average daily intakes of PUFAs, MUFAs, and SFAs were significantly higher in the cases than in the controls. The ratios of PUFAs to SFAs were 0.7 for the cases and 0.8 for the controls. The intakes of PUFAs, MUFAs, and SFAs as a percentage of total energy intake were also significantly higher in the cases than in the controls: 4.4%, 7.5%, and 6.8% of energy for the cases compared with 4.1%, 6.4%, and 5.9% of energy for the controls, respectively.

The results of the multivariate analyses are shown in **Table 3**. The odds ratios (ORs) for fat intake were adjusted for the effects of possible confounders (BMI and smoking status). When other risk factors were taken into account, the OR for the association of total fat intake with IHD was 1.08 (95% CI: 1.02, 1.14). When absolute amount of fat intake was used instead of fat intake as a percentage of energy, the OR for fat intake was also significant (OR: 1.03; 95% CI: 1.01, 1.05). In the multivariate analysis we also adjusted for a history of hypertension and found trivial effects on the association between dietary fat and IHD incidence.

We also compared 2 multivariate analyses examining the relation between the subtype of fat (PUFAs, MUFAs, and SFAs) and IHD risk (**Table 4**). Model 1 controlled for BMI, cigarette smoking, and energy intake, but not total fat intake. Model 2 controlled for the variables included in model 1 and for total fat intake. SFA intake (OR: 1.15; 95% CI: 1.02, 1.30) and MUFA intake (OR: 1.12, 95% CI: 1.01, 1.25) were both positively associated with IHD risk in model 1. In model 2, however, the associations between intakes of SFAs, PUFAs, and MUFAs and IHD risk were not significant.

To examine whether the dose-response relation between total fat intake and the risk of IHD incidence was linear, we classified total fat intake in 4 groups and calculated the OR in each group after adjustment for BMI, smoking status, and energy intake.

**TABLE 2**Mean intakes and mean percentages of energy from specific nutrients<sup>1</sup>

Nutrient	Cases (n = 108)	Controls (n = 142)
Energy (kJ)	9873.6 ± 3057.4	9302.9 ± 2646.9
Carbohydrate		
(g)	357.5 ± 109.7	354.6 ± 101.4
(% of energy)	61.2 ± 7.3	64.3 ± 7.1 <sup>2</sup>
Protein		
(g)	85.1 ± 29.6	78.0 ± 28.0
(% of energy)	14.5 ± 2.6	13.9 ± 2.7
Total fat		
(g)	60.4 ± 28.5	50.2 ± 22.3 <sup>2</sup>
(% of energy)	22.4 ± 5.9	19.9 ± 5.2 <sup>2</sup>
PUFAs		
(g)	11.8 ± 5.6	10.2 ± 4.4 <sup>3</sup>
(% of energy)	4.4 ± 1.3	4.1 ± 1.2 <sup>3</sup>
MUFAs		
(g)	20.3 ± 11.8	16.4 ± 9.4 <sup>2</sup>
(% of energy)	7.5 ± 2.9	6.4 ± 2.5 <sup>2</sup>
SFAs		
(g)	18.6 ± 10.5	15.0 ± 8.3 <sup>2</sup>
(% of energy)	6.8 ± 2.5	5.9 ± 2.2 <sup>2</sup>
P:S	0.7 ± 0.3	0.8 ± 0.3

<sup>1</sup> $\bar{x} \pm$  SD. PUFAs, polyunsaturated fatty acids; MUFAs, monounsaturated fatty acids; SFAs, saturated fatty acids; P:S, ratio of polyunsaturated to saturated fatty acids.

<sup>2,3</sup>Significantly different from cases: <sup>2</sup>P < 0.01, <sup>3</sup>P < 0.05.

**TABLE 3**

Adjusted odds ratios and 95% CIs of developing ischemic heart disease for selected risk factors from logistic regression analysis<sup>1</sup>

Variable	Odds ratio (95% CI)
Body mass index (kg/m <sup>2</sup> )	
≥25	2.26 (1.27, 4.01)
<25	1.00
Smoking status	
>1 pack/d	10.51 (3.61, 30.56)
≤1 pack/d	2.40 (1.03, 5.62)
Exsmoker	2.67 (1.09, 6.55)
Never smoked	1.00
Energy (kJ)	1.00 (1.00, 1.00)
Total fat intake (1% of energy intake)	1.08 (1.02, 1.14)

<sup>1</sup>Model included BMI (2 categories: <25 and ≥25), cigarette smoking (4 categories: never, past, ≤1 pack/d, and >1 pack/d), and energy intake.

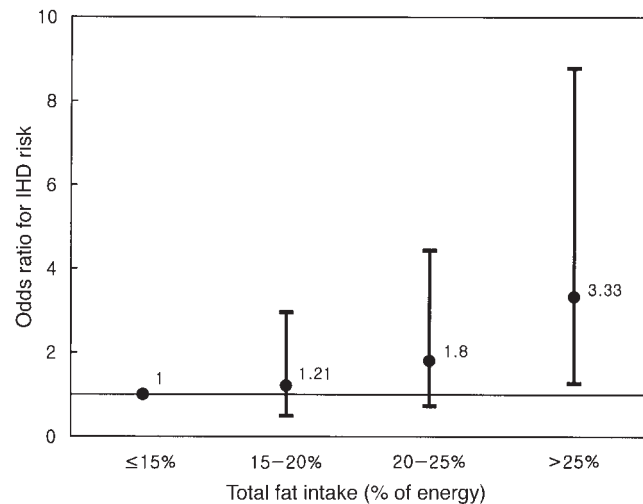
There was a trend in the association of IHD according to amount of total fat intake. The ORs were significant only for the group with a total fat intake of >25% of total energy (**Figure 1**).

We examined the data for the possibility of effect modification by age, BMI, smoking status, and history of hypertension or diabetes. We used the median values for the controls to dichotomize the distribution of BMI to examine the effect of fat intake on IHD incidence in each BMI group. The effect of fat was not significantly different between the 2 groups. Also, the effect of fat on IHD incidence was not significantly different according to smoking status or history of hypertension or diabetes.

We also compared the characteristics of 2 control subgroups (patients admitted to the ophthalmologic department or the orthopedic department). The distribution of the controls in the age, BMI, smoking status, alcohol intake, exercise, history of hypertension, and nutritional intake categories was not significantly different between the 2 control subgroups. Also, there were no significant differences for these variables when we compared each of the control subgroups with the cases.

## DISCUSSION

Our study was conducted in a university teaching hospital that serves an urban population. The cases had newly developed acute myocardial infarction confirmed by electrocardiogram and enzyme measurement and angina pectoris confirmed by coronary angiogram. Thus, the study population included only valid cases. The use of incident cases eliminated the possibility that the cases had modified their behaviors because of a previous diagnosis of IHD. The design also allowed comparison of risk factors among individuals of a homogeneous ethnic origin within



**FIGURE 1.** Adjusted odds ratios and 95% CIs for risk of ischemic heart disease (IHD) according to total fat intake.

a geographic region. Our study did have the limitations inherent in a case-control design. However, although the controls were hospital based, we carefully selected patients from the ophthalmology and orthopedic surgery departments so that it would be unlikely that the prevalence of certain risk factors was different in the controls than in the general population. To improve the recall and reliability of the information obtained, we included only subjects aged ≤75 y. Even though the cases and controls were recruited from only one hospital, the results may be generalizable to all Korean men. The mean (±SD) BMI of the controls (23.7 ± 2.9) is similar to that of healthy Korean men aged >30 y (22.8 ± 2.7) (25) and the total fat intake of the controls (19.9% of energy intake) is similar to the mean total fat intake of Koreans (19.1% of energy intake) (22).

Tobacco use, BMI, hypertension, and diabetes or glucose intolerance are important modifiable risk factors for IHD in Western countries (26–29). Our data show that tobacco use is also a significant risk factor in Korean men. We found a dose-response relation between smoking status and IHD. Patients who currently smoked >1 pack/d had higher ORs than did those who smoked ≤1 pack/d and exsmokers. BMI was also a significant risk factor for IHD in Korean men, even though the average BMI of these men was much lower than that in Western countries. Information on blood pressure in this study was based on self-reported history of diagnosed and treated hypertension. Although we had data on the patients' blood pressures after admission, we did not use this information because blood pressure may have been modified by

**TABLE 4**

Adjusted odds ratios and 95% CIs of developing ischemic heart disease for total fat and subtypes of total fat<sup>1</sup>

	Model 1			Model 2		
Total fat (% of energy)	—	—	—	1.08 (0.98, 1.18)	1.11 (0.99, 1.24)	1.08 (1.01, 1.16)
SFAs (% of energy)	1.15 (1.02, 1.30)	—	—	1.00 (0.81, 1.25)	—	—
MUFAs (% of energy)	—	1.12 (1.01, 1.25)	—	—	0.94 (0.75, 1.18)	—
PUFAs (% of energy)	—	—	1.21 (0.97, 1.50)	—	—	0.97 (0.72, 1.31)

<sup>1</sup>Model 1 included BMI (2 categories: <25 and ≥25), cigarette smoking (4 categories: never, past, ≤1 pack/d, and >1 pack/d), and energy intake. Model 2 added total fat intake to model 1. SFAs, saturated fatty acids; MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids.





treatment or by the cardiovascular event. A history of hypertension was a significant risk factor for IHD in the univariate analysis but was not included in the multivariate model because it was not a confounder. In addition, a history of diabetes mellitus was slightly higher in the cases than in the controls but was not a significant risk factor for IHD in our study.

For several decades, the debate on diet and IHD has been dominated by the classic diet-heart hypothesis. Diet is believed to be a major factor in the etiology of IHD, but considerable scientific uncertainty exists about the relation between specific dietary components and IHD risk, as well as epidemiologic doubt about the adequacy of the classic diet-heart hypothesis (30–32). The present study assessed nutritional factors that had been proved to be associated with IHD in Western countries in Korea, where dietary intake of total fat is much lower than in Western countries.

Among the controls, total fat intake was  $\approx 20\%$  of energy intake, which is lower than in Western countries (35–42% of energy intake) (1–6). Intake of PUFAs was similar to that in Western countries, but SFA intake in our study was only 6–7% of energy intake, one-half of that reported in other studies (13–18% of energy intake). The average energy composition of the diet of the controls in this study was 64.3% carbohydrates, 13.9% protein, and 19.9% total fat and was in the range of the recommendations (65%, 15%, and 20%, respectively) of the Korean Nutrition Society (22).


The relation between IHD and SFA intake has been shown consistently in numerous epidemiologic studies, such as the Honolulu Heart Program (3), the Seven Countries Study (6), the Ireland-Boston Diet-Heart Study (5), and the Israeli Ischemic Heart Disease Study (33). No significant association was found in the Western Electric Study (1), the Zutphen Study (2), or the Puerto Rico Heart Health Program (4), but, with the exception of the Western Electric Study (1), these findings had limited power because of small study sizes or inadequate dietary assessment (29, 34). In our study, the SFA intake of the cases was higher than that of the controls (6.8% compared with 5.9% of energy, respectively). Also, PUFA intake in our study was 4.4% of energy in the cases and 4.1% of energy in the controls. Total fat intake was a significant risk factor for IHD in multiple logistic regression analysis after adjustment for smoking status, BMI, and energy intake. Although the results of this study agree with those of the epidemiologic studies of Western populations, our study is one of the first to examine the association between dietary fat and IHD in an East Asian population.

To examine the relation between subtype of total fat and IHD, Ascherio et al (35) constructed a logistic regression model that controlled for energy and total fat intakes, whereas Hu et al (36) constructed a logistic regression model that controlled for energy intake only. In our study, the relations between the subtypes of fat (SFAs, MUFAs, and PUFAs) and IHD were different according to adjustment for total fat intake in the logistic regression model. That is, the subtypes of fat (especially SFAs and MUFAs) were positively associated with the risk of IHD after adjustment for energy intake only. However, these associations became non-significant after further adjustment for total fat intake. Therefore, any type of fat may confer risk of IHD in this low-fat-consuming population. Further research will be needed to determine the subtype of fat that might be influencing the risk of IHD.

In the Health Professionals Follow Up Study (35), dietary fiber intake was found to be an important confounder of the association between SFA intake and risk of IHD. Also, Pietinen et al

(37) reported that an increase in dietary fiber intake might be protective against IHD. We investigated the effect of total fat intake on risk of IHD after controlling for fiber intake and found a significant positive association (data not shown).

A similar finding was reported in China, where dietary patterns are more or less similar to those in Korea and the rate of IHD is lower than in the West. The Chinese diet has changed dramatically in the past 10 y and is more westernized than before, and the incidence of and mortality from IHD varies widely in different regions of China (8, 38). In urban areas, IHD is the leading cause of death and the mortality rate from IHD is twice as high as in rural areas. Campbell and Junshi (9) conducted an ecologic survey of dietary and mortality characteristics in 65 regions of rural China and concluded that intakes of total fat and small intakes of food of animal origin are associated with the increase in plasma cholesterol concentrations, which are associated, in turn, with the increase in the chronic degenerative disease mortality rate.

The present study showed that a moderate dietary fat intake may be a significant risk factor for IHD incidence in a population with a low fat intake. This finding suggests at least 2 explanations. First, the effect of total fat intake on IHD incidence may differ according to the distribution of total fat intake in each population. Alternatively, if the effect of fat intake on IHD is similar across populations, current guidelines that recommend a total fat intake of 30% of energy may be too high for preventing IHD in Western countries. There is a need to study these issues. For developing countries, there is a need to find the safe fat intake level for the population to prevent epidemics of IHD. 

## REFERENCES

1. Shekelle RB, Shryock AM, Paul O, et al. Diet, serum cholesterol, and death from coronary heart disease. The Western Electric Study. *N Engl J Med* 1981;304:65–70.
2. Kromhout D, Coulander CDL. Diet, prevalence and 10-year mortality from coronary heart disease in 871 middle-aged men. The Zutphen study. *Am J Epidemiol* 1984;119:733–41.
3. McGee DL, Reed DM, Yano KY, Kagan A, Tillotson J. Ten-year incidence of coronary heart disease in the Honolulu Heart Program. Relationship to nutrient intake. *Am J Epidemiol* 1984;119:667–76.
4. Garcia-Palmieri MR, Sorlie P, Tillotson J, Costas R Jr, Cordero E, Rodriguez M. Relationship of dietary intake to subsequent coronary heart disease incidence: The Puerto Rico Heart Health Program. *Am J Clin Nutr* 1980;33:1818–27.
5. Kushi LH, Lew RA, Stare FJ, et al. Diet and 20-year mortality from coronary heart disease. The Ireland-Boston Diet-Heart Study. *N Engl J Med* 1985;312:811–8.
6. Keys A, Menotti A, Karvonen MJ, et al. The diet and 15-year death rate in the Seven Countries Study. *Am J Epidemiol* 1986;124:903–15.
7. Sugano M. Characteristics of fats in Japanese diets and current recommendations. *Lipids* 1996;31:S283–6.
8. Chen J, Gao J. The Chinese Total Diet Study in 1990. Part II. Nutrients. *J AOAC Int* 1993;76:1206–11.
9. Campbell TC, Junshi C. Diet and chronic degenerative diseases: perspectives from China. *Am J Clin Nutr* 1994;59(suppl):1153S–61S.
10. Wenxun F, Parker R, Parpia B, et al. Erythrocyte fatty acids, plasma lipids, and cardiovascular disease in rural China. *Am J Clin Nutr* 1990;52:1027–36.
11. Suh I, Jee SH, Kim IS. Changing pattern of cardiovascular diseases in Korea. *Korean J Epidemiol* 1993;15:40–6.
12. Ministry of Health & Social Welfare, Republic of Korea. '95 National Nutrition Survey report. Seoul, Republic of Korea: Ministry of Health & Social Welfare, 1997.



13. Park SH, Shin GJ. Lipoprotein(a) as a risk factor for coronary heart disease—whether related with NIDDM or not. *Korean J Circ* 1996; 26:507–13.
14. Park JK, Kim HJ, Park KS, et al. The case-control study on the risk factors of cerebrovascular diseases and coronary heart diseases. *Korean J Prev Med* 1996;29:639–55.
15. Park CG, Kim YH, Suh HS, et al. Lipids and lipoprotein(a) levels in patients with coronary artery disease. *Korean J Circ* 1993;23: 634–43.
16. Jeon YD, Kim SY, Choi RK, Rhee MY, Lee HS, Yoo SW. Clinical study of risk factors in patients with acute myocardial infarction. *Korean J Circ* 1994;24:937–41.
17. Jung SA, Park SH, Shin GJ, Lee YH. The role of insulin resistance as a risk factor of coronary artery disease. *Korean J Circ* 1996;26:35–43.
18. Chae SC, Jun JE, Park WH, Kim JC, Jung TH. Plasma lipids and apolipoproteins as risk factor of ischemic heart disease. *Korean J Circ* 1991;21:229–39.
19. Kim SY, Lee YC, Cho SY. Nutrients and individual fatty acids intake patterns in the coronary artery disease patients with different degree of stenosis. *Korean J Nutr* 1997;30:976–86.
20. Lim HS, Baik IK, Lee HS, et al. Effects of the life style in patients with coronary artery disease on the serum lipid concentrations and atherosclerotic coronary lesion. *Korean J Lipidol* 1995;5:71–83.
21. Chung YS, Kim HM, Kim HS, et al. Changes in degree of coronary artery narrowing after life-style modification in angiographically documented coronary atherosclerotic patients. *Korean J Intern Med* 1995;48:181–9.
22. The Korean Nutrition Society, Republic of Korea. Recommended dietary allowances for Koreans. Seoul, Republic of Korea: The Korean Nutrition Society, 1995.
23. Lee YC, Lee HJ, Oh KW. Fatty acid composition of Korean foods. Seoul, Republic of Korea: Shin Kwang Publisher, 1995.
24. Ministry of Health & Social Welfare, Republic of Korea. Korean food composition table. Seoul, Republic of Korea: Ministry of Health & Social Welfare, 1996.
25. Jones DW, Kim JS, Andrew ME, Kim SJ, Hong YP. Body mass index and blood pressure in Korean men and women: the Korean National Blood Pressure Survey. *J Hypertens* 1994;12:1433–7.
26. Neaton JD, Wentworth D, for the MRFIT Research Group. Serum cholesterol, blood pressure, cigarette smoking and death from coronary heart disease. Overall findings and differences by age for 316,099 white men. *Arch Intern Med* 1992;152:56–64.
27. Willett WC, Green A, Stampfer MJ, et al. Relative and absolute excess risks of coronary heart disease among women who smoke cigarettes. *N Engl J Med* 1987;317:1303–9.
28. Manson JE, Stampfer MJ, Hennekens CH, Willett WC. Body weight and longevity: a reassessment. *JAMA* 1987;257:353–8.
29. Kannel WB, Vokonas PS. Primary risk factors for coronary heart disease in the elderly: the Framingham Study. In: Wenger NK, Furgberg CD, Pitt E, eds. *Coronary heart disease in the elderly*. New York: Elsevier, 1986:60–92.
30. Willett W. *Nutritional epidemiology*. New York: Oxford University Press, 1990.
31. Ascherio A, Willett WC. New directions in dietary studies of coronary heart disease. *J Nutr* 1995;125:647–55.
32. Woodward DA, Limacher MC. The impact of diet on coronary heart disease. *Clin Nutr* 1993;77:849–61.
33. Goldbourt U, Yaari S, Medalie JH. Factors predictive of long term coronary heart disease mortality among 10,059 male Israeli civil servants and municipal employees. A 23-year mortality follow-up in the Israeli Ischemic Heart Disease Study. *Cardiology* 1993;82:100–21.
34. Pietinen P, Ascherio A, Korhonen P, et al. Intake of fatty acids and risk of coronary heart disease in a cohort of Finnish men. The Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study. *Am J Epidemiol* 1997;145:876–87.
35. Ascherio A, Rimm EB, Giovannucci EL, et al. Dietary fat and risk of coronary heart disease in men: cohort follow up study in the United States. *BMJ* 1996;313:84–90.
36. Hu FB, Stampfer MJ, Manson JE, et al. Dietary fat intake and risk of coronary heart disease in women. *N Engl J Med* 1997;337:1491–9.
37. Pietinen P, Rimm EB, Korhonen P, et al. Intake of dietary fiber and risk of coronary heart disease in a cohort of Finnish men: The Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study. *Circulation* 1996;94:2720–7.
38. Khor GL. Nutrition and cardiovascular disease: an Asia Pacific perspective. *Asia Pac J Clin Nutr* 1997;6:122–42.

